of variance revealed a statistically significant difference across all four groups (control group, SCI group, naMCI group, aMCI group) \((p < 0.001)\). Whereas 8% of controls reported considerable degree of SMC, 34% of the SCI group, 31% of the naMCI group and 54% of the aMCI group reported considerable SMC. A two factor analysis of variance with the factors cognitive status (controls, SCI group, naMCI group, aMCI group) and depressive status (depressed vs. not depressed) and SMC as a dependent variable revealed that both factors were significant \((p < 0.001)\) whereas the interaction was not \((p = 0.820)\). A large proportion of patients seeking help in a memory outpatient clinic report considerable SMC, with increasing degree from cognitively healthy elderly to aMCI. Depressive status increases SMC consistently across groups with different cognitive status.


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A critical role for molecular chaperones in Alzheimer’s disease
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Background and objectives: Chaperones are one of the best examples of multifunctional proteins and their production against neurodegeneration may result from one or more of their activities in cells, perhaps in addition to their ability to inhibit fibril formation directly. Our objective was to explore if an excess of chaperone capacity in the cell can shift the equilibrium between amorphous and fibrillar aggregates and if the cells’ proteolytic machinery can more efficiently turn over the toxic proteins. The area investigated may help explain chaperone suppression of neurotoxicity.

Material and methods: Expression pattern analysis of Hsp90/70 was determined by using Western Blot and immunohistochemistry on postmortem cortical tissues from Alzheimer’s disease cases and aged matched controls.

Results: The results showed that AD samples contained significantly higher levels of the Hsp90/70 and this elevation was associated with the disease pathology. No cross reaction was observed between antibody used for Hsp90/70 (H0010/N27, respectively) and monoclonal antibody TG3. TG3 stains neuritic plaques and neurofibrillary tangles but does not significantly react with tau from normal human biopsy tissue; therefore exhibiting TG3. TG3 stains neuritic plaques and neurofibrillary tangles and this elevation was associated with the factors cognitive status (controls, SCI group, naMCI group, aMCI group) and depressive status (depressed vs. not depressed) and SMC as a dependent variable revealed that both factors were significant \((p < 0.001)\) whereas the interaction was not \((p = 0.820)\). A large proportion of patients seeking help in a memory outpatient clinic report considerable SMC, with increasing degree from cognitively healthy elderly to aMCI. Depressive status increases SMC consistently across groups with different cognitive status.


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Increased menin expression associated with neural apoptosis in the frontal cortex of SIV infected macaques
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Various types of neuronal damage have been reported to be associated with dementia complex (ADC). Human immunodeficiency virus (HIV) infected microglial/macrophage, not neuron directly in the brain. However, the damage of the neuron existed after the infection, in which the regulatory protein Tat of HIV-1 could be the critical factor. It has been recently reported that HIV-1 Tat transactivation requires menin, which hints that menin may be involved in the pathogenesis of ADC. However, the mechanism has not been elucidated completely. In this study, we report the up-regulated menin expression in the frontal cortex of SIV-infected macaques, especially in the neuronal nucleus by the double-labeling immunofluorescence, qRT-PCR and Western blot assay. The co-localization of menin and caspase3 is also observed in the frontal cortex of SIV-infected macaques. TGF-β, a cytokine associated with menin expression, is also detected increased in the frontal cortex of SIV-infected macaques. Furthermore, co-localization of SIV Tat and TGF-β appeared in increased TGF-β expressed vertebral neuron. In conclusion, these results indicate that...